Letters to the Editor . . .

PREVENTION OF TISSUE ANOXIA

A new method of preventing tissue anoxia is reported by Proger[1] and his associates of Tufts College Medical School, Boston, Mass. The method is based on their observation that under conditions of inadequate (10 per cent) oxygen pressure in the inspired air the venous blood contains 4-6 volumes per cent of available oxygen. The accompanying tissue anoxia is thus due largely to inadequate oxygen avidity of the fixed tissue cells. Theoretically, the addition of respiratory enzymes to the fixed tissues might increase oxygen consumption.

Early experiments[2] showed that intravenous, intraperitoneal or intramuscular injections of respiratory catalysts improve tissue utilization of oxygen in living animals. More detailed experiments were afterwards undertaken on both man and laboratory animals, using the most important intra-cellular respiratory pigment, cytochrome C. This respiratory catalyst was selected because of its easy isolation from the beef heart, and its chemical stability which makes it clinically utilizable. Preliminary tests showed that in its purified condition, cytochrome C is non-toxic and apparently non-antigenic for both man and laboratory animals.

Injected intravenously, in 350 mg. doses, cytochrome C is not excreted in human or canine. It is retained in effective concentration for at least five days where it is largely absorbed by the fixed tissue cells. In vitro tests show that it may increase oxygen consumption by the fixed tissues by as much as 100 per cent.

Such an increase is clinically effective. In both man and dogs the effects on the electrocardiogram produced by moderately severe anoxia (10 per cent oxygen) can be regularly prevented by previous intravenous injection of this catalyst. Patients who experienced subjective distress with this degree of anoxia were free from distress when they had been previously injected with this pigment. Cytochrome C has a moderately beneficial effect in angina pectoris but no immediate effect in acute myocardial infarction. This latter may be due to the fact that the total occlusion of the blood vessels makes it impossible for cytochrome C to reach the infarcted tissue.

Studies of certain cerebral functions have demonstrated that cytochrome C is effective in preventing many of the consequences of partial anoxia of the brain. The electro-encephalographic changes produced by 10 per cent oxygen can be largely prevented. The impairment of visual discrimination can be entirely overcome within about five minutes. Such complex cerebral functions as code transliteration can be strikingly influenced. The fatal irreversible state which usually follows severe hemorrhagic shock in dogs can be prevented when large doses of cytochrome C are injected a few hours after the hemorrhage.

From these and other experimental and clinical observations, Proger concludes that it is desirable to explore the possible clinical usefulness of cytochrome C in such conditions as Raynaud's disease, pulmonary emphysema, certain cerebral dysfunctions, and in various degenerative diseases associated with arteriosclerosis. A more detailed report of current clinical evidence is promised for the near future.[3]

REFERENCES

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3. Proger, S. and Dekaneas, D.: J. Pediat. (In press.)

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